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Inga Reynisdottir

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EXAMINER

BAUSCH, SARAE L

ART UNIT

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**Please find below and/or attached an Office communication concerning this application or proceeding.**

The time period for reply, if any, is set in the attached communication.

<b>Office Action Summary</b>	<b>Application No.</b> 10/820,226	<b>Applicant(s)</b> REYNISDOTTIR ET AL.	
	<b>Examiner</b> SARAE BAUSCH	<b>Art Unit</b> 1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

### Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

### Status

- 1) ☒ Responsive to communication(s) filed on 11 March 2008.
- 2a) ☐ This action is **FINAL**.                      2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

### Disposition of Claims

- 4) ☒ Claim(s) 53 and 54 is/are pending in the application.
- 4a) Of the above claim(s) 54 is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 53 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

### Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

### Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All    b) ☐ Some \*    c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
  2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
  3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

### Attachment(s)

- |                                                                                        |                                                                   |
|----------------------------------------------------------------------------------------|-------------------------------------------------------------------|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892)            | 4) <input type="checkbox"/> Interview Summary (PTO-413)           |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948)   | Paper No(s)/Mail Date. _____                                      |
| 3) <input checked="" type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08) | 5) <input type="checkbox"/> Notice of Informal Patent Application |
| Paper No(s)/Mail Date <u>03/06</u>                                                     | 6) <input type="checkbox"/> Other: _____                          |

### **DETAILED ACTION**

1. This action is in response to applicants correspondence mailed 03/11/2008.

#### ***Election/Restrictions***

2. Applicant's election of group I in the reply filed on 03/11/2007 is acknowledged. Because applicant did not distinctly and specifically point out the supposed errors in the restriction requirement, the election has been treated as an election without traverse (MPEP § 818.03(a)). Furthermore it is noted that the election of the combination of markers was not a species election but a restriction requirement as explained in section 4 of the restriction mailed 09/07/2007. It is noted that applicant elected only one marker, SG05S808.
3. Claim 54 withdrawn from further consideration pursuant to 37 CFR 1.142(b) as being drawn to a nonelected invention, there being no allowable generic or linking claim. Election was made **without** traverse in the reply filed on 03/11/2007.
4. Claim 53 and the marker SG05S808 are under examination.

#### ***Claim Rejections - 35 USC § 112-Description***

5. The following is a quotation of the first paragraph of 35 U.S.C. 112:  
  
The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.
6. Claim 53 is rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described

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in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The rejected claims are broadly drawn to kit for diagnosing susceptibility to type II diabetes in an individual comprising primers for nucleic acid amplification of an at risk marker of the KCHIP1 gene, wherein the primers are suitable for nucleic acid amplification of at least one at risk marker SG05S808 and markers in linkage disequilibrium. The rejected claims further encompass primers for SG05S808 in an individual and thus encompass detecting the marker SG05S808 in any species, human and non-human. The rejected claims encompass primers for any marker in linkage disequilibrium. Thus, the claims are broadly drawn a variety of nucleic acids, including any polymorphic variant of KChIP1 or any polymorphism in linkage disequilibrium that is associated with type II diabetes in any individual, human or non-human.

When the claims are analyzed in light of the specification, the instant invention encompasses nucleic acids comprising the analysis and detection of an enormous and wide variety of nucleic acid sequences. The claims are broadly drawn to a plurality of nucleic acids that are suitable for amplification of at least one risk marker of T2D, which encompasses an extremely large genus of polymorphic variants of KChIP1 gene in any species with any nucleotide content (A or G or C or T) at any position. Thus the claims encompass the detection of any of the many different nucleic acids wherein the nucleic acid sequence is correlated with an association of disease. Nucleic acids of such a large genus have not been taught by the specification.

In analyzing whether the written description requirement is met for genus claims, it is first determined whether a representative number of species have been described by their

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complete structure. The instant specification provides the sequence of SEQ ID No. 1 which corresponds to chromosome 5 of Build 33 of the GenBank database human genome sequence and includes the KChIP1 genomic DNA. The specification does not demonstrate any polymorphisms in linkage disequilibrium with SG05S808 associated with type II diabetes in any individual. The specification teaches that a polymorphism in KChIP1 gene can be an insertion or deletion of a single nucleotide, more than one nucleotide, change of at least one nucleotide, deletion of several nucleotides, insertion of several nucleotides, duplication of all or part of the gene (see pg. 56, last paragraph con't to page 57), however the specification does not teach which of these polymorphisms would be predictably associated with increase or decrease susceptibility to type II diabetes. Furthermore, no sequence or polymorphisms were described for any other species other than human.

Next, it is determined whether a representative number of species have been sufficiently described by other relevant identifying characteristics (i.e. other than nucleotide sequence, gene name, and specific polymorphic position), specific features and functional attributes that would distinguish different members of the claimed genus. In the instant case, the specification provides only the sequence of SEQ ID no. 1, which is part of chromosome 5 according to Build 33 of the GenBank database human genome sequence and an encoded amino acid sequence of SEQ ID No. 2. The specification does not provide any characteristics that would allow one to identify any particular portions or fragments or variants of the disclosed sequence that would allow for the diagnosis of type II diabetes in any subject based on detection of the non-disclosed gene or variants. Furthermore, the art discloses that there are 9 known species of the gene KChIP1 (see GeneCard pg. 8) and there are 1397 SNPs known for the KChIP1 gene (see

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GeneCard, page 9). Neither the specification nor the prior art teach an association with any of these SNPs with increased or decreased susceptibility of type II diabetes in any individual, human and non-human.

Applicants' attention is directed to the decision in *In re Shokal*, 113 USPQ 283 (CCPA 1957) wherein is stated:

It appears to be well settled that a single species can rarely, if ever, afford sufficient support for a generic claim. *In re Soll*, 25 C.C.P.A. (Patents) 1309, 97 F.2d 623, 38 USPQ 189; *In re Wahlforss et al.*, 28 C.C.P.A. (Patents) 867, 117 F.2d 270, 48 USPQ 397. The decisions do not however fix any definite number of species which will establish completion of a generic invention and it seems evident therefrom that such number will vary, depending on the circumstances of particular cases. Thus, in the case of small genus such as the halogens, consisting of four species, a reduction to practice of three, or perhaps even two, might serve to complete the generic invention, while in the case of a genus comprising hundreds of species, a considerably larger number of reductions to practice would probably be necessary.

In the instant application, because of the lack of a representative genus of SNP analysis regarding of the KCHIP1 gene other than the marker SG05S808 in humans, or any other polymorphisms in linkage disequilibrium corresponding to KChIP1, one of skill in the art cannot envision the detailed chemical structure of the nucleic acid encompassed by the claimed methods, regardless of the complexity or simplicity of the method of isolation. Adequate written description requires more than a mere statement that such nucleic acids are part of the invention and reference to a potential method for identification. The particular nucleic acids are themselves required.

In conclusion, the limited information provided regarding the nucleic acids of the claimed methods is not deemed sufficient to reasonably convey to one skilled in the art that Applicant is in possession of kit comprises primers for identifying a risk marker or markers in linkage disequilibrium associated with type II diabetes in any individual, human or non-human.

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Thus, having considered the breadth of the claims and the provisions of the specification, it is concluded that the specification does not provide adequate written description for the claims.

***Claim Rejections - 35 USC § 112-Scope of Enablement***

7. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

8. Claims 1, 3-4, 50-53, 57-59, and 61-63 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a kit comprising primers for nucleic acid amplification of the risk marker SG05S808 does not reasonably provide enablement for the a kit for diagnosing susceptibility or predisposition to type II diabetes in an individual comprising primers for nucleic acid amplification of an at risk marker of the KChIP1 gene, wherein the primers are suitable for nucleic acid amplification of at least one at risk marker selected from SG05S808 and markers in linkage disequilibrium. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make or use the invention commensurate in scope with these claims.

Factors to be considered in determining whether a disclosure meets the enablement requirement of 35 USC 112, first paragraph, have been described by the court in *In re Wands*, 8 USPQ2d 1400 (CA FC 1988). *Wands* states at page 1404,

“Factors to be considered in determining whether a disclosure would require undue experimentation have been summarized by the board in *Ex parte Forman*. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples,

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(4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims.”

The nature of the invention and the breadth of the claims

The claims are drawn to a kit for the diagnosis of type II diabetes in an individual comprising primers for nucleic acid amplification of a risk marker, SG05S808 or markers in linkage disequilibrium.

The rejected claims encompass analysis of any individual, including human and non-human.

The rejected claims encompass analysis of increased and decreased susceptibility or predisposition to type II diabetes.

The rejected claims encompass primers for nucleic acid amplification of risk marker SG05S808 or markers in linkage disequilibrium with SG05S808 in any species.

The nature of the claims requires the knowledge of a correlation between the risk markers in KChIP1 nucleic acid and type II diabetes in an individual. The claims require the knowledge of a correlation between primers for detection of a risk markers that corresponds to SG05S808 in any species with diagnosing a predisposition to type II diabetes.

Guidance in the Specification and Working Examples

The specification asserts that polymorphisms in a KChIP1 nucleic acid indicate a susceptibility to type II diabetes (see pg. 2, lines 30-32 and page 3 lines 1-5). The specification asserts that methods of diagnosing type II diabetes by at risk haplotypes and at risk haplotype embraces one or a combination of haplotypes over the KChIP1 gene and can be characterized by



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the presence of at least one SNP as described in table 13 (see pg. 9, lines 5-15). The specification teaches that a polymorphism in KChIP1 gene can be an insertion or deletion of a single nucleotide, more than one nucleotide, change of at least one nucleotide, deletion of several nucleotides, insertion of several nucleotides, duplication of all or part of the gene (see pg. 56, last paragraph con't to page 57), however the specification does not teach how to use this large genus of polymorphisms to diagnose an increase or decreased predisposition type II diabetes. The specification does not teach an association between SG05S808 and type II diabetes in any individual. Thus, the specification does not provide any guidance on how to use the primers for nucleic acid amplification of an at risk marker or markers in linkage disequilibrium of the KChIP1 gene. Furthermore, the recitation of the marker SG05S808 does not limit the primers to risk markers in a specific nucleic acid sequence and could encompass the same or different positions in different species for example human, dog, hamster, etc..

The specification teaches isolating a nucleic acid from a sample and determining polymorphisms in a nucleic acid which include PCR, LCR, nested PCR (see page 21, lines 9-17). Although determination of alleles is routine in the art, predictably correlating a marker to predisposition of type II diabetes in any human or non-human individual is unpredictable and the specification does not predictably teach risk markers of SG05S808 or markers in linkage disequilibrium associated with susceptibility to type II diabetes (T2D) in any human or non-human individual and thus does not teach how to use primers for nucleic acid amplification of an at risk marker of the KChIP1 gene for diagnosing a susceptibility or predisposition to T2D.

The specification does not demonstrates a working example of marker SG05S808 and its association with T2D

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The specification does not teach analysis of any non-human individuals. The specification does not demonstrate that markers in linkage disequilibrium with SG05S807 is predictably associated with increased or decreased susceptibility to T2D.

The unpredictability of the art, the state of the prior art, and level of skill in the art

While the state of the art and level of skill in the art with regard to detection of a polymorphism in a known gene sequence is high, the level of unpredictability in associating any particular polymorphism with a phenotype is even higher. The level of unpredictability is demonstrated by the prior art, the post filing art, and the instant specification.

The prior art does not teach any association between marker SG05S808 predisposition to type II diabetes. The prior art does not teach any association between any marker in linkage disequilibrium with SG05S808 and predisposition to risk for type II diabetes in any individual.

It is unpredictable as to whether or not a marker that corresponds to SG05S808 exists in any non-human organisms, and whether or not this marker in such a sequence in any other organism would be predictive of the risk of type II diabetes mellitus. For example, Mummidi et al. (2000). Mummidi et al. teaches the sequence analysis of the CC chemokine receptor 5 (CCR5) gene in humans and non-primates. Notably, the reference teaches that the substantial interspecies sequence variation is observed for the cis-regulatory regions of the CCR5 gene (p. 18949, right column, 1<sup>st</sup> full paragraph). Thus it is entirely unpredictable as to whether or not any polymorphism, including marker SG05S808 would be associated with type II diabetes in any other non-human organism.

Furthermore, the post filing art teaches the unpredictability of determining an association in different ethnical groups with any polymorphism KChIP1 with type II diabetes. Barroso et al.

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(Diabetic Medicine 2005, 22 :517-535) teach that evidence of genetic component of type II diabetes is a result of underlying differences in genes, with different frequencies of predisposing alleles present in different populations (see page 517, 2<sup>nd</sup> column, 1<sup>st</sup> paragraph). Barroso et al. teach that diabetes is not one but many diseases with a common phenotype and this makes the genetic studies harder to undertake as the definition of the disease can vary between studies. Barroso et al. teach that the problem is that it has been difficult to clinically distinguish each of the different categories of diabetes (see page 518, 2<sup>nd</sup> column, last paragraph). Barroso et al. teach that the understanding of the molecular bases of complex disease is still in its infancy and the identities of genes predisposing to many complex disorders, not just type II diabetes, remain elusive. Barroso et al. teach that several features of complex diseases are at the root of this – variable age of onset, reduced penetrance (the presence of different degrees of disease severity), locus and allelic heterogeneity as well as the presence of phenocopies means that many genes in multiple biological pathways are likely to interact with environment to increase or decrease susceptibility, many genes are expected to have a role in complex diseases each with a small effect (see pg. 523, 2<sup>nd</sup> column, 2<sup>nd</sup> and 3<sup>rd</sup> full paragraph). Barroso et al. further teaches that few associations have been replication in additional populations (see page 525, 1<sup>st</sup> column, 1<sup>st</sup> paragraph). Therefore, Barroso et al. teaches that unpredictability of associating allele frequencies and polymorphism detection in type II diabetes in any ethnic group. Therefore, Barrosos et al. teaches the unpredictability of associating allele frequencies and polymorphism detection in type II diabetes risk.

Additionally, the prior art teaches that there are many parameters that need to be evaluated prior to using a genetic test to determine a disease and that these parameters yield gaps in information that are needed to complete a thorough screening of a genetic test.

Additionally, Ionnidis (Plost Med, 2005, 2(8):e124) teach that most published research findings are false. Ionnidis et al. teach that ill-founded strategy of claiming conclusive research finding solely on the basis of a single study assed by formal statistical significance represented and summarized by p values (see pg. 0696, 2<sup>nd</sup> column, 1<sup>st</sup> full para.) Ionnidis et al. teach that research findings are likely to be true that in fields that undertake large studies, such as randomized controlled trials (several thousand subjects randomized) than in small studies such as sample sizes 100 fold or smaller (see pg. 0697, 3<sup>rd</sup> column, 2<sup>nd</sup> full para.) Ionnidis et al. teaches that what matters is the totality of evidence and that statistical significance of a single study only gives a partial picture (see pg. 0701, 1<sup>st</sup> column). Additionally, Hattersley et al. (Lancet, 2005, vol 366, pp. 1315-1323) teaches that the key quality in an association study is sample size (see page 1318, 2<sup>nd</sup> column, 1<sup>st</sup> full paragraph). Hattersley et al. teach that sample sizes of thousands are needed to detect variants that are common but have low relative risk and teach that allelic odds ratio of 1.1 to 2.0 requires the number of controls to be in thousands (see page 1318, 2<sup>nd</sup> column, 1<sup>st</sup> full paragraph and table 3). Hattersley et al. teach that apparent studies in identifying interesting associations with studies much smaller than implied by table 3 (in the thousands) might suggest that calculations are too pessimistic and small initial studies rarely find the correct result and even when they do they are likely to overestimate the true effect size (see page 1318, 1<sup>st</sup> column, 1<sup>st</sup> full paragraph). Hattersley et al. further teaches that emphasis has been on the need for greater stringency in the association studies in order to prove a given association and

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suggest a p value of  $5 \times 10^{-8}$ , however arguments from Bayesian perspective suggest that  $5 \times 10^{-5}$  should be sufficient to constrain the false discovery rate. It is further relevant to point out that Hegele (2002) teaches the general unpredictability in associating any genotype with a phenotype. Hegele teaches that often initial reports of an association are followed by reports of non-replication and refutation (p.1058, right col., lns.24-30). Hegele provides a table indicating some desirable attributes for genetic association studies (p.1060), and includes choosing an appropriate significance threshold (see 'Minimized type 1 error (FP)') and replication of results in independent samples (see 'Replication'). Additionally, Hegele teaches the desirability of a likely functional consequence predicted by a known or putative functional domain.

#### Quantity of Experimentation

Given the lack of guidance in the specification with regard to association of marker SG05S808 or markers in linkage disequilibrium and increase OR decrease likelihood of diagnosis of type II diabetes mellitus along with the evidence in the art that demonstrates the unpredictability of using primers for nucleic acid amplification of an at risk marker of the KChIP1 gene and type II diabetes. Thus, the quantity of experimentation in this area is extremely large as associating the marker SG05S808 and markers in linkage disequilibrium is unpredictable. The skilled artisan would have to perform an extremely large study and include different populations and familial studies each polymorphism and each haplotype of KChIP1 to determine if in fact there was either an association between the polymorphism any individuals and type II diabetes mellitus. The results of such a study are clearly unpredictable as evidence by the post filing art (which reflects the current state of the art) and the teachings in the specification with regard to lack of analysis of the risk marker SG05S808. Barroso et al.

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(Diabetic Medicine 2005, 22 :517-535) teach that evidence of genetic component of type II diabetes is a result of underlying differences in genes, with different frequencies of predisposing alleles present in different populations (see page 517, 2<sup>nd</sup> column, 1<sup>st</sup> paragraph). Barroso et al. teach that diabetes is not one but many diseases with a common phenotype and this makes the genetic studies harder to undertake as the definition of the disease can vary between studies. Barroso et al. further teaches that few associations have been replication in additional populations (see page 525, 1<sup>st</sup> column, 1<sup>st</sup> paragraph).

In the instant case, it would be unpredictable as to whether or not primers for nucleic acid amplification of the marker SG05S808 or markers in linkage disequilibrium would be able to be used to determine a decreased or increased likelihood of susceptibility of type II diabetes in any individual. Thus, it is unpredictable to associate any polymorphism or haplotype in the KChI P1 nucleic acid with T2D.

In order to practice the invention as broadly as it is claimed, the skilled artisan would have to determine polymorphisms in KChIP1 that are in linkage disequilibrium with SG05S808 as well as analysis of SG05S808, which encompass the same or different position in different species in each individual in each species and then determine which polymorphism are associated and would detect increased and decreased susceptibility in type II diabetes mellitus in large study of more than a thousand individual. The skilled artisan would than have to determine which primers would amplify and would be suitable for detection of the markers. The skilled artisan would have to perform an extremely large amount of trial and error analysis in a large study to determine if such polymorphisms would predictably determine a susceptibility to type II diabetes mellitus. Given the lack of guidance in the specification and the post filing art with

respect to accurately testing genetic diseases, such analysis is replete with unpredictable experimentation and is considered undue.

***Claim Rejections - 35 USC § 102***

9. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

10. Claim 53 is rejected under 35 U.S.C. 102(b) as being anticipated by Rhodes et al. (WO 00/31133).

Rhodes et al. teach PCIP nucleic acid molecules as hybridization probe and PCR primers for amplification of PCIP nucleic acid molecules. Rhodes et al. teach using all or portions of SEQ ID No. 1 as a hybridization probe and primers (see pg. 34, 35 lines 1-11 and page 38, last paragraph). Rhodes et al. teach SEQ ID No. 1 is human KChIP1 (see figure 1 and table 1). Additionally Rhodes teaches PCR and primers of SEQ ID no. 1 and thus teaches primers for nucleic acid amplification of an at risk marker of KChIP1 nucleic acid (see pg. 98 lines 1-8 and pg. 101-102). Rhode further teaches a diagnostic kit (see page 40, lines 10-12).

***Conclusion***

11. No claims are allowable.

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Any inquiry concerning this communication or earlier communications from the examiner should be directed to Sarae Bausch whose telephone number is (571) 272-2912. The examiner can normally be reached on M-F 9am-5pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on (571) 272-0735. The fax phone number for the organization where this application or proceeding is assigned is (571) 273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at (866) 217-9197 (toll-free).

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

Patent applicants with problems or questions regarding electronic images that can be viewed in the Patent Application Information Retrieval system (PAIR) can now contact the USPTO's Patent Electronic Business Center (Patent EBC) for assistance. Representatives are available to answer your questions daily from 6 am to midnight (EST). The toll free number is (866) 217-9197. When calling please have your application serial or patent number, the type of document you are having an image problem with, the number of pages and the specific nature of the problem. The Patent Electronic Business Center will notify applicants of the resolution of the problem within 5-7 business days. Applicants can also check PAIR to confirm that the problem has been corrected. The USPTO's Patent Electronic Business Center is a complete service center supporting all patent business on the Internet. The USPTO's PAIR system provides Internet-based access to patent application status and history information. It also enables applicants to view the scanned images of their own application file folder(s) as well as general patent information available to the public.

For all other customer support, please call the USPTO Call Center (UCC) at 800-786-9199.

/Sarae Bausch/  
Primary Examiner, Art Unit 1634